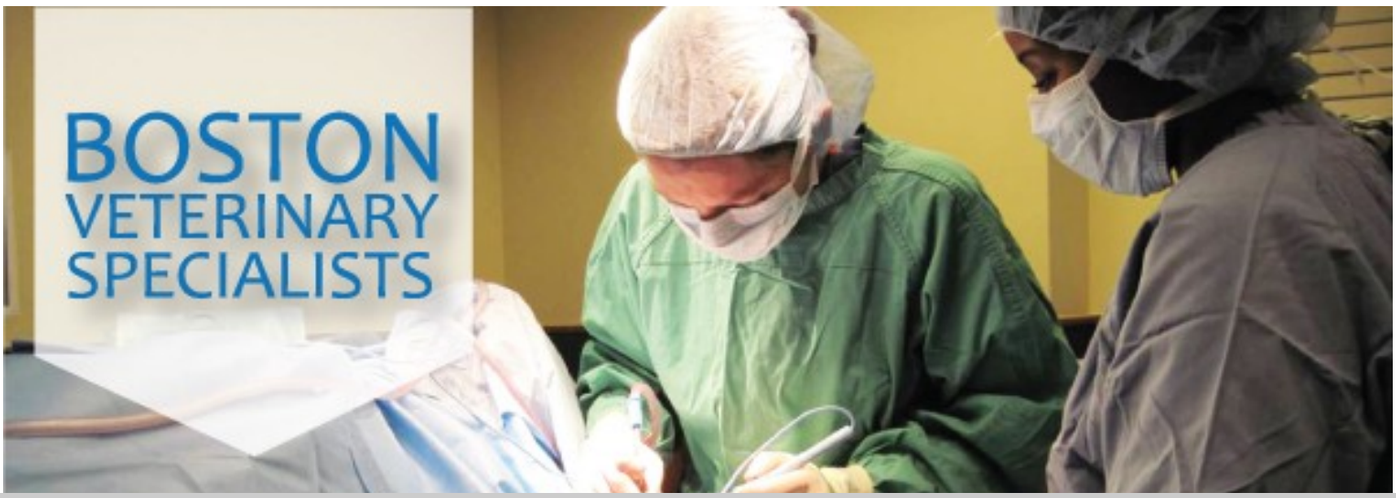

Monthly Update **May 2013**
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Endocrine Emergencies Part 2: Diabetic-ketoacidosis (DKA) and Thyrotoxicosis aka "Thyroid Storm"

Diabetic-ketoacidosis

Diabetic-ketoacidosis: Diabetic-ketoacidosis (DKA) is a severe form of Diabetes Mellitus (DM) characterized by hyperglycemia, acidosis and ketosis. DKA is typically due to a combination of low endogenous insulin levels (i.e.: secondary to DM) as well as elevated counter-regulatory hormone levels (i.e.: glucagon, cortisol, catecholamines, growth hormone). This increase in counter-regulatory hormones is only possible in the presence of a concurrent disease, so it is of utmost importance to search for the underlying disease when a diagnosis of DKA has been made. In dogs, the most commonly reported concurrent diseases are urinary tract infections, pancreatitis, and Cushing's disease. Cats commonly have hepatic lipidosis, pancreatitis, infections (viral, bacterial) or cancer as their secondary disease process.

Pathogenesis

In diabetics, lack of insulin results in decreased glucose uptake by the cells. This results in the Krebs cycle halting completely due to lack of pyruvate, which is a breakdown product of glycolysis. With the Krebs cycle in standstill, there is build up of acetyl-coenzyme-A (a-coA) in the cells as this typically enters the Krebs cycle with pyruvate. In addition, secondary to increased glucagon, the fatty acid concentration increases within cells, which then undergo beta-oxidation to form a-coA. The combination of these 2 conditions, decreased insulin and decreased glucagon, leads to overall increased a-coA in the cells, which drives the production of ketone bodies. The two dominant ketoacids are acetoacetate and beta-hydroxybutyrate (BHA). Acetone is a neutral ketone body that is also formed.

Risk Factors

Patients with DKA tend to be middle age, with the mean age for dogs and cats being around 8-9 years old, and there is no breed or sex predilection with DKA. Physical exam findings are often non-specific and could be attributed to chronic unmanaged diabetes, the concurrent disease or the acute DKA event. These patients are often polyuric and polydypsic, dehydrated, lethargic, and anorexic and many show signs of vomiting. Cranial abdominal organomegally may be appreciated, as well as a poor hair coat and mental dullness. Dogs may have cataracts as a complication of DM and cats may develop icterus.

Diagnosis

On routine blood work (CBC, biochemistry panel), 100% of these patients will have hyperglycemia (unless they were recently treated with insulin). Dogs will often have elevated serum ALP, while roughly 50% of dogs in one study had elevations in their ALT, AST and cholesterol. Cats will commonly have elevated ALT and cholesterol, while azotemia is a more common clinical finding in cats than dogs. The CBC in both dogs and cats is largely non-specific with a non-regenerative anemia, left shift neutrophilia, and thrombocytosis. Cats, however, may develop Heinz bodies (which is correlated with plasma BHA levels).

These patients will often have a venous pH < 7.35 on a blood gas analyzer, however, some animals can be diabetic and ketotic, without developing acidosis.

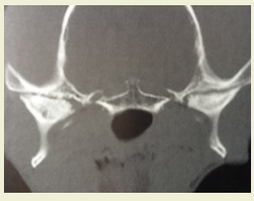
Continued next page

CT Scan Diagnostic

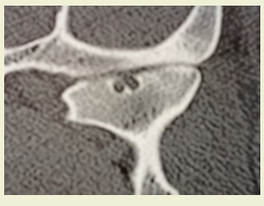
Each month we will show an anatomic area that CT studies have provided us with better diagnostic than digital radiography. This month is TMJ joints.

TMJ joints are difficult to visualize on radiographs. There can be dislocations, fractures, DJD secondary to trauma, benign cysts, OCD lesions. CT scans provide much better visualization of these injuries and diseases.

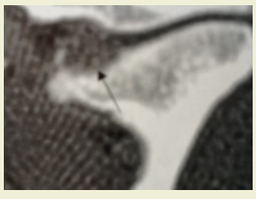
Imaging features of three dogs with masticatory pain.

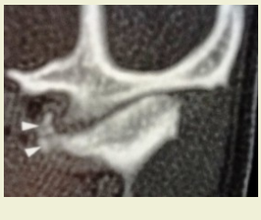


Bilateral irregular bony joint margins of the TMJ and adjacent sclerosis, consistent with DJD.



Subchondral bone cyst in the mandibular condyle.





Subchondral bone defect (arrow) and corresponding free bone fragments in the medial TMJ (arrowheads). The last two are are consistent with osteochondrosis.

Ketones

Checking for ketones can be done via multiple methodologies. The urine reagent test strips utilize the nitroprusside reaction and can be used with either urine or plasma. The only ketone bodies tested for via the nitroprusside reaction are acetoacetate and acetone, not BHA. This means that some ketoacidotic patients may be overlooked if their serum acetoacetate hasn't reached renal threshold and isn't being excreted in the urine yet. In addition, concurrent use of some drugs (N-acetylcysteine, captopril and penicillamine) can interfere with the test and cause false positive results. Previously, the use of hydrogen peroxide (H_2O_2) was thought to increase the sensitivity of the nitroprusside test, as H_2O_2 acts as a catalyst for the conversion of beta-hydroxybutyrate back to acetoacetate. This effect, however, is too minimal to cause any significant increase in sensitivity and shouldn't be relied upon.

Electrolytes

Patients with DKA commonly have multiple electrolyte disturbances. Their potassium is often normal to elevated on presentation, which can be secondary to dehydration, decreased renal excretion, lack of insulin and decreased insulin function, as well as cellular shifting secondary to acidemia. With rehydration, true hypokalemia (total body potassium depletion) becomes apparent. Insulin therapy, as well as vomiting, inappetence, anorexia, and osmotic diuresis may further exacerbate hypokalemia. Potassium supplementation is required when contemplating beginning insulin therapy (dose: ranges from 0.05mEq/kg/hour to Kmax: 0.5mEq/kg/hr) with frequent (anywhere from SID to TID) electrolyte rechecks.

Initial blood work also often shows hyponatremia, however this is not a true hyponatremia and can be explained by the concept of pseudo-hyponatremia. In pseudo-hyponatremia, excessive glucose pulls fluid osmotically into the vascular space, which dilutes the concentration of sodium causing an artificial hyponatremia. Sodium should be correct for hyperglycemia by the simple formula:

- For every 100mg/dL the glucose is above normal glucose level (around 100mg/dL), there is a 2 mEq/L decrease in sodium concentration. This value needs to be added to the measured [Na] to obtain the "corrected sodium" level.

The corrected Na reflects the true hydration status of the patient and allows the clinician to understand the true state of water balance in their patient. Similarly to Addisonian patients, it is of utmost importance to choose a fluid that matches the sodium concentration of the patient during the resuscitation phase to avoid osmotic myelinolysis syndrome. It's also important to monitor electrolytes frequently (q 6-8hours) during the initial stabilization period. As a patient's electrolyte imbalances and hydration status normalize the frequency of monitoring can be reduced.

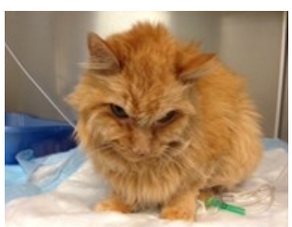
Other potential electrolyte abnormalities that are commonly noted in DKA patients include hypophosphatemia, hypomagnesemia, hypochloremia and hypocalcemia. Low phosphorous is clinically significant due to the potential for marked systemic hemolysis, as well as the potential to induce seizures. Hypomagnesemia is associated with difficulties in normalizing potassium levels due to its cofactor status for the Na-K ATPase pumps in the kidney. Both of these electrolytes should be supplemented if low and regularly monitored (at least once daily) to ensure normalization (potassium] phosphate CRI: 0.03-0.12mMol/kg/hr; magnesium sulfate CRI: 1mEq/kg/day). Ionized hypocalcemia should be confirmed via a blood gas analyzer and should be supplemented if the patient shows associated clinical signs: muscle weakness, trembling/tremoring, or unresponsive hypotension.

The quest for an underlying disease process

It is important to realize that DKA occurs secondary to another disease process that is responsible for the production of counter-regulatory hormones. The search for this process in imperative. Common second diseases and diagnostics are listed below.

- Pancreatitis- serum cPL level, abdominal ultrasound
- Pyelonephritis- urinalysis and culture via cystocentesis or sterile catheter (culture even if urinalysis looks quiet; DM produces dilute urine which can hide signs of UTI)
- Cushing's disease: ACTH/low-dose dex AFTER discharge from hospital, abdominal ultrasound
- Aspiration pneumonia: chest radiographs, endotracheal or trans-tracheal wash if indicated
- Region specific infections: 4DX snap, Tick-borne disease PCR, CBC with path review,
- Prostatitis/pyelonephritis (if intact): rectal exam, abdominal radiographs, abdominal ultrasound
- Neoplasia: thorough physical examination, chest radiographs, abdominal ultrasound.

Treatment



Similarly to Addison's disease, the most important priority in patients with DKA is to restore intravascular volume, restore cardiovascular stability and reverse shock. As with Addisonian patients, during the initial fluid resuscitation time, attempts should be made to match the sodium level of the fluids with that of the patient to avoid rapid changes in sodium concentration. The effects of IV fluid therapy are multifactorial. They not only act to restore intravascular volume, but because of improved renal perfusion, glucose will be eliminated and acid base status can improve even before insulin therapy commences.

Before considering insulin therapy it is vital that all three of the following criteria are met:

- cardiovascular stability
- normal serum potassium levels
- restoration of urine output to the expected polyuria seen in stable DM

This can often take 6-8 hours from presentation and thus insulin is not considered an emergent treatment for DKA but rather a necessary second step once the patient is stabilized to clear ketones and restore a positive energy balance.

Overall Prognosis
Before an owner can make an informed decision of whether to treat their pet with DKA they need to be educated that DM is a life-long disease requiring injections twice a day, and the initial regulation can be intensive and costly. In addition, dog-owners need to know that the development of cataracts is almost certain in dogs. The overall prognosis for patients with DKA is very good, with 70% of dogs/cats treated for DKA surviving to discharge. Prognosis is affected by the underlying concurrent disease, with pyelonephritis typically being easier to regulate than Cushing's disease, which requires more intensive insulin therapy and longer hospital stays. The median hospitalization time for a dog or cat with DKA is roughly 5-6 days, with a 7% recurrence rate in dogs and a 40% recurrence rate in cats.

Thyroid Storm

Thyrotoxicosis is a term used to describe any condition where there's an excessive amount of circulating thyroid hormone or a rapid increase in circulating thyroid hormones. These patients usually have undiagnosed thyroid gland hyperfunction (thyroid adenomas in cats, thyroid carcinomas in dogs). Rarely other causes could lead to a thyroid storm such as overdosing of exogenous thyroid hormone (i.e. accidental ingestion a bottle of Thyroxin), abrupt cessation of anti-thyroid medication, thyroid gland surgery, post-radioactive iodine therapy, or rigorous thyroid palpation. There are also non-thyroidal illnesses that can lead to thyrotoxicosis such as increased sympathetic stimulation or increased cellular response to thyroid hormones, which can be found in sepsis, overwhelming infections, hypoxemia, or hypovolemia.

Clinical signs
The most common clinical signs are tachypnea and tachycardia. The cardinal sign in cats is panting. These signs may be secondary to circulating thyroid hormone, but may also be associated with an underlying or secondary problem, such as congestive heart failure or hypertension. Thyrotoxic patients who develop secondary heart failure due to their over-circulating thyroid hormone actually have a good prognosis as this condition can be reversible. Their clinical signs mirror primary CHF with dyspnea, murmurs/gallop rhythms and pulmonary crackles. Signs from hypertension may include retinopathies with retinal hemorrhage, retinal detachment or even epistaxis. Unfortunately, a common clinical finding is death, which illustrates how fragile these patients truly are.

Diagnosis
Thyroid storm/thyrotoxicosis requires a clinical diagnosis. It is based on clinical signs, historical precipitating events as well as the presence of the potential underlying disease process, such as hyperthyroidism or thyroid carcinoma. It may be possible to identify the toxicosis via elevated total T4 or free T4, however, this should not be performed at the risk of the patient's life if they are in distress, especially since the results will not be available prior to beginning treatment.

Treatment
Treatment is aimed at controlling the following four areas:

- Counteract peripheral effects of thyroid hormones
- Systemic support to reverse the effects of thyroid hormone in the body
- Reduce production of new thyroid hormone
- Remove precipitating factor

Counteract peripheral effects
Hypersensitivity to adrenergic stimulation is the main peripheral effect of overproduction of thyroid hormone. This is achieved with beta-blockers. In this scenario, the amount of beta-blocker needed is the lowest dose that achieves desired physiologic end points. Because atenolol is oral, it is generally best to start with the low-end of the dose and recheck a heart rate, respiratory rate and reevaluate the patient after 2 hours of dosing. Desired end-points are a heart rate less than 200bpm, cessation of panting and a blood pressure between 120-180mmHg, systolic. Excessive administration of beta-blockers can result in hypokalemia and CHF so always titrate this drug carefully.

Systemic support
Because diagnostics such as chest radiographs and blood-draws can be life-ending in these fragile patients, it is best to treat the symptoms of their distress while waiting for onset of beta blockade. This means providing oxygen support if they are tachypneic, a cool environment if they are hyperthermic and correcting electrolyte imbalances (if electrolytes levels can safely be obtained). Because many hyperthyroid cats are hypokalemic at baseline and beta blockers can reduce serum potassium levels further, supplementing KCl at 20mEq/L is often suggested.

Reduce production of thyroid hormone
For feline patients this means starting methimazole therapy as soon as the patient is stable enough to eat, for canine patients this may mean a more extensive work up to diagnose and treat thyroid carcinoma.

Remove the precipitating factor
At least part of the precipitating factor is transport and evaluation at a veterinary hospital since this is a stressful event for these patients. Placement of the patient in a cool, dim, quiet environment will often alleviate the signs of their crisis. Often the white noise of a circulating oxygen cage is soothing to hyperthyroid cats although due to risks for hyperthermia only O2 cages that have the ability to regulate temperature should be used. These patients should be handled for only short periods as infrequently as possible. This means diagnostics may need to be obtained in a step-wise fashion to honor their fragility.

Outcome
In humans, this is a rare disease but one with a high mortality rate in the emergency rooms. In veterinary medicine, the true incidence and mortality rate is unknown but early recognition and aggressive treatment is paramount to survival. The overall prognosis depends on the underlying cause and response to stabilization and therapy.

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Tech Tip

ANESTHETIC MONITORS-UNDERSTANDING THEIR USE AND LIMITATIONS**

Performing anesthesia is a task that most veterinary technicians undertake on a daily basis. Intra-operative monitoring is imperative for optimizing all anesthetic procedures. In addition to allowing informed, flexible and well-timed responses to changes in the patient's status, it can also serve as a database for comparison prior to subsequent anesthetic episodes. A variety of equipment is available to monitor the patient's physiologic parameters, including but not limited to stethoscopes, blood pressure monitors, electrocardiograph (ECG) tracings, pulse oximeters, end-tidal carbon dioxide monitors, and temperature probes. Is one monitor better than the others? One must first consider the overall effects of general anesthesia before evaluating monitors that would be ideal for assessing patients under general anesthesia. It is well known that inhalant anesthetics are potent respiratory depressants. They are potent vasodilators, readily causing hypotension at increased levels of anesthesia. Decreased cardiac output, central nervous depression, and muscle relaxation are also direct effects of inhalant anesthetics. With this information in mind, let's examine the various monitoring modalities and exactly what they tell us about the anesthetized patient.

Electrocardiography (ECG)
ECG monitoring is commonplace during general anesthesia. It is important to ensure good contact of leads to skin by either using ECG paste or alcohol when placing ECG leads. Avoid wetting large areas of the skin and direct contact with the table. Exact lead locations are not as important as ensuring that all waves are present (even if they are inverted). The P-wave represents atrial depolarization. The QRS-wave represents ventricular depolarization. The T- wave indicates ventricular re-polarization. It is important to realize that an ECG tracing does not provide information about chamber size, or how efficiently the heart is ejecting blood. Therefore, the ECG should be used strictly for the detection of dysrhythmias during the peri-anesthetic period.

Induction agents and disease processes may predispose patients to cardiac arrhythmias. Other potential causes of cardiac arrhythmias may include an inadequate or excessive anesthetic depth, pain, hypoxia, hypercapnia, heart or lung disease, and traumatic myocarditis. Electrolyte imbalances and acidosis may also be a source of cardiac arrhythmias. It is not always necessary to treat arrhythmias unless they are causing adverse affects to the patient. Bradycardia is commonplace in patients undergoing general anesthesia and can be defined as a heart rate of <80 beats per minute in a small dog, or <60 beats per minute in large dogs. In cats, bradycardia is defined as <100 beats per minute. There are numerous causes for bradycardia, which may include drug side-effects, excessive vagal tone, hypertension, hyperkalemia, uremia, hypothermia, increased intracranial pressure, profound hypoxemia, and deep-level inhalants, among others. Tachycardia is defined as >180 beats per minute in a dog, and >200 beats per minute in cats. Tachycardic states can lead to hypotension. There are many causes of tachycardia, which may include but are not limited to, drugs, an inadequate plane of anesthesia, hyperthermia, anaphylactic reactions, hypovolemia, early-stage hypercarbia, and numerous disease states.

Pitfalls associated with interpretation of ECG tracings can include lead mal-positioning due to broken clips or loose connections to the monitor. Electrical interference caused by cauteryo or other operating room equipment can also be problematic. Rate inaccuracies can occur based on the size of the waveform, resulting in either double-counting or non-counting issues. Patient motion secondary to shivering or increased respiratory rates can cause blurred or erratic tracings. As a final caveat, electrical activity is often the last aspect to completely disappear prior to the pronouncement of death.

Blood Pressure

It is important to realize that all patients experience some degree of hypotension during general anesthesia, and if the patient has pre-existing conditions that decrease blood pressure, hypotension will be exacerbated during anesthesia. Normal arterial blood pressure values for canines are systolic 110-119mmHg & diastolic 55-110mmHg, and for felines, systolic 120- 170mmHg & diastolic 70-120mmHg.

Although direct blood pressure monitoring is considered the "gold standard", it is highly impractical when it comes to routine blood pressure monitoring in most privately-owned veterinary facilities due to the advanced skill level required to place arterial catheters and the need for 24-hour care. Therefore, only indirect methods of blood pressure monitoring will be discussed. There are 2 methods to measure blood pressure indirectly - either by using a Doppler or an oscillometric device (e.g., Dinamap, Cardell, petMAP). Regardless of the method used, selection of the correct sized blood pressure cuff is imperative for providing the most accurate results. The width of the cuff should extend 40% around the circumference of the limb. When the cuff is determined to be too small, the next wider size should be selected. In cats, it is acceptable to use a cuff that is only 30% of the circumference of the limb. The cuff should be snug, but not too tight. It is acceptable to use a piece of tape to keep it from becoming dislodged during cuff inflation. Selection of an inappropriate cuff size is the most common source of errors. If the cuff is too narrow or too loose, the reading will be falsely high. If the cuff is too wide or too tight, the reading will be falsely low. Acceptable cuff locations include the forelimb, tail and hindlimbs, where the areas proximal to the carpus and tarsus work best. The ventral tail is a good choice in cats and short-legged breeds such as the Bassett hound and Dachshund.

Oscillometric methods detect intracuff changes caused by the pulse wave. They calculate the systolic, diastolic and mean arterial pressure (MAP) as well as the heart rate. They frequently can be programmed to obtain readings at various time intervals (e.g., once per minute, per hour.)

Doppler methods use a 'return-to-flow' principle to detect the systolic blood pressure. Doppler measurements are most accurate when the systolic blood pressure is within normal limits and when the patient has good peripheral perfusion. In cats it is hypothesized that the resultant reading probably represents the MAP, therefore a correction factor of 14mmHg is added to the reading to more accurately reflect actual feline systolic pressures. Because the 'white coat' phenomenon has been well documented in humans, the patient should be calm and as well-acclimated as possible to avoid an inadvertent false diagnosis of hypertension or hypotension. Be warned that a Doppler can mistake heavy respirations for blood flow. Profound arrhythmias, hypothermia, patient motion, low batteries, and electrical interference can also impede obtaining good readings.

There are drawbacks associated with indirect methods of blood pressure monitoring. In general they all tend to underestimate the actual blood pressure, and all work best when the MAP is between 60-100mmHg. Patient movement, smaller patient size (<5.0kg), cold or vasoconstricted patients, or patients with short-legs or excessive skin will all adversely affect results. Additionally, measurements may be difficult to obtain in patients with limb edema.

Pulse Oximetry

Pulse oximeters provide continuous and non-invasive monitoring of pulse and an estimate of arterial hemoglobin saturation (SpO2), but do not provide data on the amount (partial pressure) of oxygen in arterial blood, as dissolved in plasma (PaO2). Pulse oximeters can be used on the lip, tongue, ear pinna, prepuce, vulva, toe web or digits, metacarpus, tail, rectal mucosa or flank skin folds. If a skin-fold site is selected it should ideally be hairless, non-pigmented, and fairly thin-skinned (but not overly so). In large animals consider using the nostril/nasal septum as well.

There are 5 main types of hemoglobin: oxyhemoglobin, reduced hemoglobin (deoxyhemoglobin), methemoglobin, carboxyhemoglobin, and fetal hemoglobin. Since 95% of oxygen delivery to tissues is by oxyhemoglobin, saturation is of high clinical significance. Not all types of hemoglobin are capable of transporting oxygen, and as such are termed "dysfunctional hemoglobins." The presence of other light-absorbing types of hemoglobin such as methemoglobin and carboxyhemoglobin will cause the pulse oximeter to overestimate arterial oxygen saturation. Conversely, extraneous blood-borne dyes (such as methylene blue) are known to potentially lower SpO2 readings to 85%, regardless of the true saturation value. Pigmented substances such as bilirubin lipids (hyperbilirubinemia) may also affect arterial blood light absorption and alter SpO2 values. Other causes for erroneous SpO2 values include severe anemia or hemodilution. Moreover, the pulse oximeter may display an SpO2 reading of 100%, in spite of the considerable decrease in arterial blood oxygen content secondary to low hemoglobin values.

Further pitfalls of pulse oximetry use include erroneous and unreliable results or potential complete loss of function when peripheral pulsations are reduced or absent, as in the case of hypotension, hypothermia or hypovolemia. Other conditions that can contribute to unreliable pulse oximeter readings include arrhythmias and tachycardia, increased venous pulsations (e.g., right heart failure, tricuspid regurgitation, etc.), and movement artifacts (e.g., shivering.) Erroneous pulse oximeter readings may also occur when using certain Xenon arc surgery lights (resulting in an SpO2 reading of 100% and a pulse rate of 180-225), without the probe being attached to a patient!

Finally, beware the pulse oximeter is surrounded by controversy in regards to its use as a monitoring device-it is either prized or despised. This is due, in part, to the oxyhemoglobin dissociation curve, which describes the non-linear relationship between PaO2 and SpO2. For example; patients breathing 100% oxygen may have a PaO2 that is 5 times greater than the SpO2 (e.g., PaO2 = 500 mmHg: SpO2 = 100%). Since the oxyhemoglobin dissociation curve is sigmoid shaped, the hemoglobin saturation

would demonstrate only a very slight increase - going from 98% to 100%. Pulse oximeters are most beneficial when evaluating desaturation, such as when the reading drops to below 90%, which corresponds with a PaO2 that is less than 60mmHg. Pulse oximeters are most accurate within 2% to 6%, and within the 80% to 100 percentile.

Carbon Dioxide

End-tidal carbon dioxide (ETCO2) is the result of expired gases from the alveoli. End-tidal carbon dioxide analysis can be used to help assess acid/base status as well as the adequacy of patient ventilation in a variety of clinical situations. An abrupt decrease in ETCO2 can be an early and reliable indication of an impending cardiovascular collapse or cardiac arrest. Consequently, ETCO2 production can be used to assess the effectiveness of cardio-pulmonary- cerebral-resuscitation (CPCR) techniques since delivery of carbon dioxide from the lungs requires blood flow, cellular metabolism, and alveolar ventilation.

Capnometers and capnographs monitor ETCO2 by evaluating samples of the patient's exhaled gases taken from the anesthetic circuit via an adapter placed on the end of the patient's endotracheal tube. This adapter must be placed precisely at the end of the patient's nose to eliminate excessive dead space and prevent rebreathing of carbon dioxide. Capnometers provide only minimum and maximum ETCO2 values, while capnographs provide a graphic display of exhaled carbon dioxide as each breath is taken. Diagnosing abnormalities in ventilation or anesthetic circuit function are easier using the graphical data provided by a capnograph.

Normal ETCO2 values are 35-45mmHg. Under normal circumstances, ETCO2 typically underestimates the arterial carbon dioxide partial pressure (PaCO2) by a clinically insignificant 2-5mmHg. End tidal carbon dioxide values above 45mmHg indicate inadequate ventilation, necessitating ventilatory assistance via manual or mechanical means. Conversely, by allowing modest increases in ETCO2 (up to 50mmHg) the anesthetist can bolster arterial blood pressure via endogenous catecholamine release. Nonetheless, the highest ETCO2 permissible should be 60mmHg.

There are caveats to ETCO2 monitoring: Esophageal intubation, occlusion of the endotracheal tube, inadequate seal on the endotracheal tube, anesthetic circuit dysfunction/disconnects, moisture within the sampling line, hyperventilation, or respiratory and/or cardiac arrest are all potential causes of failure to detect carbon dioxide. Elevated ETCO2 levels may occur as a result of hypoventilation due to airway obstruction, pneumothorax, body positioning, or lung disease, or during periods of acutely increased metabolism (e.g., thyroid storm, or catecholamine release). Significant disparities between PaCO2 and ETCO2 indicate an inefficiency of gas exchange (e.g., dead space ventilation), which may be secondary to pulmonary embolism, thromboembolism, decreased cardiac output, or perhaps as a result of mechanical ventilation (intermittent positive pressure ventilation.) Explanations for elevated ETCO2 and inspiratory carbon dioxide may include anesthetic machine malfunction (e.g., malfunctioning valves within the breathing circuit), unsuitable fresh gas flow rates (e.g., non- rebreathing circuits), or exhausted carbon dioxide granules. Therefore, end-tidal carbon dioxide is best analyzed in conjunction with an arterial blood gas sample to yield the most complete status of respiratory function.

Temperature

Hypothermia is not only one of the most common anesthetic complications, but also the easiest to document without special equipment. The hypothalamus closely regulates core body temperature. However, this regulation can be impaired in pediatric and geriatric patients, lean breeds, and those with organ failure, large wounds or infections. Almost all anesthetized or sedated patients will lose body heat under general anesthesia, with the exception of adult Nordic breeds (i.e., Samoyed, Siberian husky, Alaskan malamute), which can actually become hyperthermic. Small patients are at the greatest risk, due in large part due to their small body- surface-to-mass ratio. Hypothermia is exacerbated in prolonged surgical procedures, especially those which expose open body cavities or use cold irrigation solutions. Hypothermia-induced bradycardia is typically non-responsive to anticholinergics. Hypothermia contributes to delayed drug metabolism and decreased hepatic metabolism, resulting in prolonged recovery and potential drug toxicity. Clotting times can be prolonged due to impaired platelet function and hemoconcentration with sludging. Hypothermia also suppresses immune function and may lead to increased infection rates.

Obviously, prevention is key when addressing hypothermia. Re-warming should be considered when the patient temperature drops to < 97.6^{o} F. There are a variety of ways to maintain an envelope of warm air around peri-operative patients. Convection-type warm air devices (e.g., BAIR Huggers®) are the most effective, followed by circulating warm water blankets. At least 60% of the body surface area must be in contact with the external heat source for re-warming efforts to be most effective. If latex gloves or bottles of warm water are to be used for smaller patients, it is essential that they are initially warmed to a temperature of <107o F and removed once they cool to the temperature of the patient. Commercially available wire electric heating-pads and heat lamps have been associated with uneven heating, thermal injury and/or electrocution and should be avoided.

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